Clinical Medicine

A Practical Approach to Improving Pain Control in Cancer Patients

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Despite a wealth of recent articles, many patients with cancer pain continue to suffer needlessly. The satisfactory treatment of cancer pain requires a variety of practical management strategies. Practicing physicians need a wider understanding of both the basic principles of analgesic therapy and the pharmacologic features of analgesics. Certain analgesics are best not used in cancer care. The use of pharmacologic adjuncts may lessen overall narcotic requirements and side effects. The appropriate use of alternative therapies can dramatically improve the quality of patients' overall survival.

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Although many patients with cancer remain pain free in the early stages of their disease, about half eventually suffer pain.[1] In terminal cancer, pain is a problem in 75% of patients.[2] Experts estimate that despite a wealth of information on pain management, 25% of all cancer patients die without adequate pain control.[3]

Illustrative Case

A 42-year-old woman with obstructive jaundice was found at an operation to have inoperable carcinoma of the pancreas, and palliative biliary decompression was done. Postoperatively the patient suffered severe pain in the anterior abdomen that radiated through to the back. While in hospital, the pain medication administered was meperidine hydrochloride (Demerol) to be given on an every-four-to-six-hour basis. Following discharge, an acetaminophen-codeine mixture was prescribed, but only on an "as-needed" basis. The patient, who was already concerned about addiction, was informed that constipation, mental changes and tolerance might all be associated with the regular use of pain medication. When the family reported persisting pain, they were told stronger drugs should be "kept in reserve" for the time that the patient was "terminally ill." For the next three months, the patient suffered episodic strong pain, especially when awaking during the night. In the last month of life, the patient was admitted to hospital with cachexia and a bedsore. Although still able to swallow, at this point the patient was given large doses of narcotics intramuscularly, resulting in drowsiness and confusion. Because the family believed the patient's disorientation was an unavoidable effect of medication, she remained isolated and uncommunicative for her last weeks.

This case shows many of the common errors in cancer pain management. With an understanding of the principles of analgesic therapy in patients with cancer, however, and the proper use of currently available analgesics, most such patients can have reasonable pain control without oversedation even in the last phases of illness.

General Principles of Cancer Pain Management

Recent expert advisory committees on the management of severe chronic pain in patients with cancer have formulated realistic goals for cancer pain management (Table 1).[4,5] A first tenet is to establish an accurate etiologic diagnosis of any individual cancer patient's pain. Studies have shown that about 75% of cancer pain is associated with direct tumor involvement, while 20% will be related to cancer therapy, and the remaining 5% of patients may be suffering pain unrelated to cancer or its therapy.[3] Because cancer patients will often have many pains, a clear idea as to the cause of each individual pain is an essential step in control because different causes may suggest specific therapies. For example, pain due to direct tumor infiltration of bone will often respond well to palliative irradiation, whereas painful breast or prostate metastases may regress dramatically with appropriate hormone therapy. It is also important to remember that not all cancer pain is responsive to analgesic agents. Conventional analgesics may not help pains that are due to nervous tissue destruction, such as dysesthesia and causalgia, whereas steroids and the tricyclic antidepressants may be beneficial.[1]

There are several important general principles regarding the use of individual analgesic agents.[1,3,6] First, a sufficient dose of analgesic must be given to relieve pain. Patients who are denied an adequate dose of analgesic become preoccupied with pain and turn into "clock watchers," anticipating the arrival of the next medication dose. This fear and anxiety may actually augment analgesic requirements. Such patients may erroneously be seen by staff as demanding unreasonable amounts of narcotic and possibly displaying addictive behavior. Second, oral analgesics should be used for as long as possible because this allows the patient a mobile life-style. In addition, many terminally ill cachectic patients have less muscle mass, and parenteral injections can result in severe pain and limit mobility. Parenteral therapy should be used only when a patient cannot take medication via the oral or rectal route. Third, each dose of analgesic medication should be given on a prophylactic basis before the effects of the previous dose have worn off. The "as-needed" administration of analgesics not only results in suffering but can actually encourage the development of tolerance because pain is harder to control once it has recurred. Fourth, analgesic dosing must be tailored to each patient's needs and periodically reassessed and retitrated. For instance, increasing somnolence in a patient receiving a stable dose of narcotic may

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indicate that the dosage is now too high. This may be due to a reduced analgesic requirement, which often follows the initial control of severe pain, or may reflect a gradual decline in hepatic or renal elimination. Regular dosage reviews will also ensure that analgesics are adjusted appropriately if new side effects develop or pain worsens.

All strong analgesics have predictable side effects that should be anticipated.[1,3] Nausea, vomiting and constipation are probably the commonest side effects of narcotic therapy. Constipation is best treated prophylactically by keeping a patient well hydrated, active and on a regular regimen of stool softeners and laxatives. One such effective program consists of docusate sodium (dioctyl sodium sulfosuccinate [Colace]), 100 mg by mouth twice a day, combined with senna (Senokot S). Nausea and vomiting are often noted with the initial administration of narcotic analgesics due to stimulation of the chemoreceptor trigger zone in the medulla. An antihistamine such as dimenhydrinate (Gravol [Canada]; Dramamine, comparable US product), 50 mg every four hours, or a phenothiazine such as prochlorperazine (Stemetil [Canada]; Compazine, comparable US product), 5 to 10 mg every four hours, may be given a half hour before the narcotic. Antiemetics can often be withdrawn in the first few weeks of therapy because narcotic-associated nausea frequently decreases with time. Metoclopramide hydrochloride (Maxeran [Canada]; Reglan, comparable US product), 10 mg every four hours, may be especially useful when nausea is accompanied by dyspepsia. This antiemetic has the added benefit of not producing such phenothiazine side effects as sedation or a dry mouth. Respiratory depression is rarely a problem with narcotic therapy given orally because the plasma concentration of narcotic necessary to suppress respiratory drive seems to be higher than the concentration required for analgesia in most patients.[7] In addition, cancer pain itself is a major respiratory stimulus. Should respiratory depression inadvertently occur, however, naloxone hydrochloride (Narcan) is the narcotic antagonist of choice and may be given intravenously (IV), intramuscularly (IM) or subcutaneously.[1] The usual initial adult dose is 0.4 mg. If given in smaller doses of 0.1 to 0.2 mg IV at two- to three-minute intervals, it is often possible to restore respiratory function without counteracting the narcotic analgesic effect or precipitating withdrawal symptoms. Other narcotic side effects that may be seen are suppression of the cough reflex, bladder spasms and urinary retention.

There is no place for placebo therapy in cancer care.[1] About a third of patients with cancer may show a transient response to the administration of inert substances that they and their therapists believe will relieve pain. Endorphins may be involved in such placebo analgesic effects. Placebo responses do not indicate that a person's pain has a psychological basis or that the patient is exaggerating the reporting of pain. In fact, the use of placebos often provides no clinical information and results in patient anger and mistrust.

Current Analgesics for Cancer Pain

With an understanding of the basic principles of analgesic therapy in cancer patients, a simple analgesic ladder may be constructed with only three levels (Figure 1).[8]

The first level consists of aspirin (acetylsalicylic acid) or aspirin-equivalent preparations. The analgesic effect of aspirin is often taken for granted due to its ready availability, but in double-blind trials, 600 mg of aspirin has been as effective as 60 mg of codeine for the relief of moderate cancer pain.[9] Both the therapeutic and toxic effects of aspirin are related to free drug concentration. Because there is extensive protein binding of aspirin, the free drug concentration is directly related to the serum albumin level. Cachectic patients with advanced cancer often have hypoalbuminemia and may require their aspirin dosage to be adjusted accordingly. Tinnitus is a common symptom heralding early aspirin toxicity. Patients should reduce their dosage when it is noted.

TABLE 1.—*Principles of Analgesic Therapy in Patients With Cancer*

- Establish an etiologic diagnosis for each pain
- Remember—not all cancer pain is responsive to analgesia
- Use adequate analgesic doses
- Use oral preparations whenever possible
- Administer analgesics prophylactically to prevent pain—no PRN orders
- Titrate analgesic doses individually for adequate control
- Anticipate side effects—constipation, nausea
- Never use placebos

PRN=*pro re nata*, "as circumstances require"

Progression	Agent	Alternatives
Nonnarcotic	ASA	Acetaminophen Naproxen Ibuprofen
↓		
Weak narcotic	Codeine	Oxycodone HCl
↓		
Strong narcotic	Morphine	Hydromorphone HCl Levorphanol tartrate Anileridine Methadone HCl

Figure 1.—The diagram shows the analgesic ladder for control of cancer pain. ASA = aspirin (acetysalicylic acid), HCl = hydrochloride

Acetaminophen is a reasonable substitute when the use of aspirin is contraindicated. On a milligram-for-milligram basis, acetaminophen is equivalent to aspirin in antipyretic and analgesic effects.[10] Acetaminophen, however, lacks the anti-inflammatory and antiprostaglandin effects of aspirin and should not be substituted if these are required.

Included as alternatives at the first level of the analgesic ladder are the wide variety of nonsteroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen (Motrin, for instance) and naproxen (Naprosyn). Like aspirin, these agents interfere with prostaglandin metabolism and may be beneficial in some cases of metastatic bone pain when osseous tumor deposits induce bone reabsorption by a prostaglandin mechanism. In general, despite pharmaceutical claims, all of the NSAIDs share aspirin's side effects, including gastric intolerance and the production to some degree of a qualitative platelet defect. Recent attention has also focused on the nephrotoxicity of these drugs, especially in older patients with compromised renal function.[11] Accordingly, these agents should be used cautiously in patients with kidney disease, ulcerative process, thrombocytopenia or any underlying qualitative platelet defect. Initial gastric intolerance may be reduced by taking the medication with food. For intractable heartburn, an alternative is the concomitant use of cimetidine (Tagamet), 300 mg by mouth three times a day, as required. Physicians should use the NSAID with which they have the most experience.

The second level of the analgesic ladder consists of weak narcotic agents of which codeine is an archetypal drug. The usual codeine dose for moderate pain is 30 to 60 mg.[9] While the dosing of morphine sulfate and other strong narcotics can

be increased almost indefinitely, nonnarcotics and weak narcotics tend to have a clinical "ceiling effect" after two to three dose increments where side effects increase proportionately more than analgesia.[8] With these medications, if pain control is not achieved within two or three adjustments, there is little point in further increases, and the patient should move up to the next rung of the analgesic ladder. Codeine is commonly included with aspirin or acetaminophen for additive effect. Popular combinations are aspirin with 30 mg of codeine or acetaminophen with 30 mg of codeine (such as Tylenol #3). The codeine equivalent oxycodone is a semisynthetic morphinelike narcotic agonist metabolized by similar pathways as codeine. Oxycodone hydrochloride is commonly prescribed in combination with aspirin (Percodan) or acetaminophen (Percocet).

The last rung of the analgesic ladder consists of morphine sulfate or equivalent agents. Because most patients with significant cancer pain will ultimately advance up the analgesic ladder, morphine has tended to be the "gold standard" against which other narcotic agents are compared.[1,3,8]

While a combination of morphine elixir, alcohol, cocaine and chloroform water (Brompton's cocktail) has been used in the past, recent practice is to use morphine solutions as single agents. Many patients objected to the bite of the alcohol in the combined preparation, and controlled trials showed cocaine added no analgesic effect, but could produce restlessness and hallucinations.[12] Commercial morphine solutions are available in a wide variety of concentrations including 1, 5, 10 and 20 mg per ml. For patients who prefer pills, morphine tablets are also available in various strengths including 5, 10 and 25 mg. In addition, a slow-release morphine tablet with prolonged duration of action (up to 12 hours) has recently been released in strengths of 30, 60 and 100 mg.[13]

When starting oral morphine therapy, the initial dose is usually 5 to 15 mg of solution. With oral morphine therapy, patients begin on an every-four-hour schedule and the next dose is given before the beneficial effect of the previous one has worn off. If pain is consistently relieved after individual doses but returns in less than four hours, the dose will have to be increased. Similarly, a dosage increment is obviously required when the initial starting dose does not consistently relieve pain. With oral morphine therapy, the usual increment is 50% of the first dose at the end of 24 to 48 hours when pain is not 90% controlled. If initial dosing controls pain but also results in significant somnolence, the next dose should be titrated downward by 25% to 50%. If titrating the dose upwards results in initial pain control that decreases with time, the dosing interval may be decreased. In practice, however, it is seldom necessary to use a shorter regimen than every four hours with administration of morphine orally. When first commencing therapy, if pain is not relieved after one or two analgesic increments of 50% to 100% or by decreasing the dose interval, consideration must be given to the possibility of narcotic-nonresponsive pain, as may be seen with degenerative nerve damage or bone destruction.

Careful assessment of total dosage every day or two during the first week of therapy is important. Once an adequate maintenance dose of oral morphine is established, individual patient requirements will often remain stable over long periods of time. Dosage escalation, however, may be required over a number of months, especially during the terminal phase of illness. Although Twycross found that more than 90% of patients with cancer would have their pain controlled with an oral morphine dose of 30 mg or less given every four hours, some individual patients with severe metastatic bone disease may require in excess of 2,000 mg a day.[4] While gradual increases in dose requirements with time may be due to the development of some degree of tolerance, sharp increases should always raise the possibility of progression of the malignant process.

TABLE 2.—*Checklist for Oral Morphine Sulfate Therapy*

Have I determined an appropriate starting dose?
Have I discussed drug side effects?
Have I discussed tolerance and dependence?
Have I prescribed an antiemetic?
Have I prescribed a laxative?
Have I explained dosage escalation if pain persists?
Has appropriate follow-up assessment been arranged?

Initially it is very important to assess and deal with patients' prejudices regarding pain and the use of analgesics. Tolerance and dependence should be discussed in a realistic fashion.[4] Many patients have strong pharmacologic prejudices and fears that, commonly voiced, include "if I use the pain medication now, it won't work when I really need it," or "if I start taking these pills on a regular basis, I'll soon be hooked on them." Patients must be reassured regarding the fallacies of such beliefs. In addition, simply showing that the prophylactic use of analgesics conserves strength and energy will often ensure compliance and avoid "as needed" dosing. Table 2 is a checklist for oral morphine therapy.

Strong analgesics that may be used as morphine alternatives include hydromorphone hydrochloride (Dilaudid), levorphanol tartrate (Levo-Dromoran), anileridine (Leritine [Canada]) and methadone hydrochloride.[14] Hydromorphone is a morphine derivative that is much more soluble than the parent compound, although the half-life is slightly shorter (four hours).[14] This property has allowed the development of a high-potency form of the medication (Dilaudid-HP) that at 10 mg per ml is especially useful in cachectic patients requiring parenteral injections. It is our impression that the oral use of hydromorphone may possibly be associated with less nausea and constipation than morphine, although this has not been tested in clinical trials. This drug is our first choice in patients who have difficulty with oral morphine preparations. Levorphanol has a longer duration of action than morphine, often providing relief for as long as six hours (Table 3). Anileridine is similar in structure to meperidine, with a shorter half-life than morphine when given IM (two to four hours). Methadone is a synthetic molecule that has attracted considerable attention as a maintenance agent for heroin addicts. Methadone analgesia usually lasts from six to eight hours and there is no clear ceiling effect. When taken orally on a regular basis, methadone differs from morphine in several ways, including greater solubility with more predictable absorption and a longer half-life (more than 24 hours in many patients). When methadone therapy is first commenced, plasma concentrations often do not reach a steady state for four to five days, and cumulative effects with respiratory depression and hallucinations can occur. Great care must be taken when using this drug, especially in debilitated elderly patients or those with hepatic or renal dysfunction.[15]

Diamorphine (heroin) has recently been extensively publicized by the lay press and has been legalized for use in cancer patients in Canada.[16] Claims made on behalf of heroin versus

TABLE 3.—*Narcotic Comparison Chart*

Pharmacology and Pharmacokinetics	*Morphine Sulfate*	*Codeine*	*Hydromorphone HCl*	*Levorphanol Tartrate*	*Anileridine*	*Methadone HCl*
Possible routes of administration . .	IM,PO,SC,IV	IM,PO,SC	IM,PO,SC,IV	IM,PO,SC,IV	IM,PO,SC	IM,PO,SC
Time to peak action, minutes	15	15-30	15-30	15-30	15	30-60
Duration of action, hours	4-5	4-6	4-5	4-5	2-3	3-5
Half-life, hours	2-3.5	3	2-3	12-16	. . .	15-30
IM equivalent dose, mg*	10	120	1.5	2	30	10
Oral equivalent dose, mg*	20-30	200	7.5	4	50	20
Oral:parenteral ratio	High 1:2	High 3:5	Low 1:5	Low 1:2	High 3:5	High 1:2

HCl=hydrochloride, IM=intramuscularly, IV=intravenously, PO=by mouth, SC=subcutaneously

*Equivalent to giving 10 mg morphine sulfate IM.

morphine include less nausea and constipation, increased mental alertness and greater solubility resulting in less painful intramuscular administration because a smaller injection volume can be used. Other than for increased solubility, these claims have not been substantiated in a number of well-conducted clinical trials. This is not surprising because pharmacologic studies have shown that in humans, heroin given orally is converted to morphine, actually providing only 80% of an equivalent oral dose.[17]

Analgesic Agents to Be Avoided

Whereas no individual analgesic is absolutely contraindicated in patients with cancer, some drugs are best avoided. Propoxyphene napsylate (Darvon-N) has been noted in several controlled trials to be less effective than aspirin.[18] Adverse effects seen with therapeutic doses of this agent include nausea and vomiting and central nervous system (CNS) side effects such as dizziness and drowsiness. In our opinion, there is little indication for the use of this drug in patients with cancer. Pentazocine (Talwin) is a weak morphine antagonist that may precipitate analgesic withdrawal if given with other narcotic analgesics. In addition, this agent has a relatively short duration of action and may produce unpleasant side effects in many patients, including nausea, vomiting, blurred vision, drowsiness and hallucinations.[19] Because 10% of patients may experience such troublesome CNS side effects, the use of this analgesic is also best avoided in patients with cancer.

Meperidine hydrochloride (Demerol) is undoubtedly the analgesic most frequently misused in cancer patients.[1,3] Meperidine is a synthetic narcotic analgesic with atropinelike effects. Unfortunately, many physicians remain unaware of meperidine's short half-life of only two to three hours.[20] Because it is most frequently prescribed on an every-four-to-six-hour basis, inadequate pain control results. The short half-life along with poor absorption when given orally limit the use of meperidine for pain control in cancer patients. In addition, if oral doses of more than 200 mg every three hours are given, there is an increased incidence of toxic CNS effects including convulsions due to the accumulation of the metabolite normeperidine.[8]

Parenteral Narcotic Therapy

Towards the end of many cancer patients' lives, parenteral administration of narcotic therapy becomes necessary. Morphine sulfate can be given parenterally by a variety of methods, including bolus injection (IV or IM), continuous drip (IV or subcutaneous) or by the epidural route. If a patient is in hospital, morphine may be given intravenously when oral therapy is no longer possible. Our protocol for a continuous morphine drip is provided in Table 4.

TABLE 4.—*Protocol for Morphine Sulfate Drip*

- Administer drug in a suitable intravenous (IV) solution and volume considering patients' 24-hour IV fluid requirements, such as 1,000 ml dextrose 5% in water
- Base the dose of morphine on previous 24-hour requirements; for instance, 20 mg given subcutaneously every 4 hours would convert to 120 mg per 24 hours at a rate of 5 mg per hour.
- Remember to reduce dose by 30% to 50% when converting from the oral or rectal routes
- Change the dose in 5-mg-per-hour increments as required and check blood pressure and respiratory rate regularly
- Do not add other ingredients to the infusion; they may be incompatible with morphine, and changes in infusion rate will result in changes in the administered dose of all ingredients

In some hospitals, the epidural administration of morphine is available and may offer some advantages, especially in those patients requiring such large morphine doses that a clouded sensorium results.[21] A number of possible complications have been noted with epidural therapy, including obstructed, broken or accidentally removed catheters and local infections including meningitis. In the future, the use of implantable delivery systems may overcome such problems. For the best analgesic results, it appears that an epidural catheter should be placed in relation to the dermatomes principally involved with the pain. Theoretically, delayed respiratory depression may be seen as long as 24 hours following each morphine dose. Intense pruritus and urinary retention have also been problems. For these reasons, this form of therapy is currently often reserved for those patients in whom conventional methods of analgesia have failed.

Pharmacologic Adjuncts to Analgesic Therapy

Phenothiazines have long been used as analgesic adjuncts in combination with traditional narcotic therapy.[1,3] We use prochlorperazine in a dose of 5 to 10 mg given at bedtime to increase the effect of morphine. Initially, patients may be more sedated with such combination therapy, but using phenothiazines in this fashion often allows the reduction of total narcotic dose and lessens overall narcotic side effects. Phenothiazines are probably most useful when a combined sedative-antiemetic effect is desired. When purely anxiolytic or sedative effects are required, the benzodiazepines are preferable as they lack the autonomic side effects seen with phenothiazines. Physicians should use the benzodiazepine preparation with which they have the most clinical experience.

The tricyclic antidepressants have proved useful adjuncts in depressed patients. It has been noted that these agents in-

hibit the presynaptic reuptake of serotonin. This may explain their beneficial effect in neurogenically mediated pain because a variety of serotonin-dependent neuronal pathways are thought to exert inhibitory effects on pain transmission.[22] The tricyclic antidepressants have long half-lives and it is often unnecessary to give full therapeutic doses in patients who are also taking narcotics. Amitriptyline hydrochloride (such as Elavil), 25 to 75 mg taken at bedtime, will increase the chance of a good night's sleep, improve daytime mood and energy and may enhance analgesia.

The benefits of steroid therapy in terminally ill patients are frequently ignored.[1,3] Steroids are useful in those patients who have compressive or closed-space syndromes including brain or liver metastases, nerve or spinal cord compression and superior vena cava syndrome. Steroids are also helpful with painful boney metastases and tumor hypercalcemia. Patients with advanced carcinomatosis who have no medical contraindications to steroid therapy, such as peptic ulcer or hypertension, will often experience improved appetite, reduced incidence of fever and an enhanced sense of well-being when maintained on glucocorticoid therapy.[23] A typical regimen might consist of an initial prednisone dose of 1 to 2 mg per kg tapered over a period of two to three weeks to a maintenance dose of 10 to 15 mg a day.

Other Therapeutic Measures

Nerve blocks are frequently underused in patients with cancer.[24] With metastatic involvement in the region supplied by the celiac plexus, as may occur with carcinoma of the stomach, pancreas or hypernephroma, celiac plexus block often results in gratifying palliation. This procedure can be done percutaneously on an outpatient basis. Patients are seldom left completely pain free, but the severity and biting nature of the pain is often reduced. Lumbar sympathetic blocks may be similarly effective with rectal, bladder or pelvic tumors involving the lumbosacral plexus that result in deep lancinating pain often referred to the anus. Cervical-thoracic sympathetic blocks may also be useful in some circumstances involving the upper extremities and neck. The benefits of individual blocks may not persist for more than a few months, but if initially successful, they can often be repeated.

There are a number of ablative neurosurgical procedures including various rhizotomies, percutaneous chordotomy and transsphenoidal hypophysectomy.[25] The discussion of these procedures is beyond the scope of this article. Because many of these operations result in significant life-long morbidity, they are usually used when conventional methods of pain control have proved unsatisfactory.

In addition, a wide variety of noninvasive adjunctive measures can be used in cancer pain control. These include such techniques as relaxation therapy, guided imagery, hypnotism, biofeedback, acupuncture and transcutaneous electric nerve stimulation.[26,27] For moderate pain, many of these modalities have been valuable adjuncts to drug therapy. Because none are uniformly suitable or available, such programs must be tailored to each patient's needs. At the very least, most physicians have access to audiocassettes that teach relaxation therapy using guided imagery.

Postscript

Using the principles outlined in this article, our patient's history could be rewritten as follows:

A 42-year-old woman with obstructive jaundice was found at a surgical procedure to have an inoperable cancer of the pancreas, and palliative biliary decompression was done, along with a prophylactic celiac plexus block. Postoperatively, the use of analgesics was thoroughly discussed with the patient and family. The patient was discharged on a regimen of acetaminophen and codeine combined, to be taken regularly on an every-four-hour basis. The patient was also given an antiemetic and was started on a program of stool softeners and laxatives to prevent constipation. After a month's duration, when pain increased, the therapy was switched to oral morphine sulfate, again taken around-the-clock on an every-four-hour basis. The morphine dose was escalated as required over the next month to keep pace with any change in pain. After three months, the patient had a repeat celiac plexus block with good palliation, such that the total morphine dose could be reduced by 30%. In the last two weeks of life, she was admitted to hospital with increasing cachexia and a bedsore, unable to manage at home. At this time, the patient's therapy was switched to hydromorphone suppositories given on an around-the-clock basis, alternating with pills when tolerated. The patient died after three weeks in hospital, able to remain in communication with family and loved ones, and having experienced good pain control.

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